

# **PUBLIC HEALTH ASSESSMENT**

## **FORMER WEST-CAP FACILITY**

*An evaluation of Occupational Exposures to  
On-Site Soils and Soil Gas*

**Tucson International Airport  
Tucson, Arizona**

**CERCLIS #AZ0980737530**



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## LIST OF ACRONYMS AND ABBREVIATIONS

ADHS	Arizona Department of Health Services
ADEQ	Arizona Department of Environmental Quality
ATSDR	Agency for Toxic Substances and Disease Registry
COC	chemical of concern
MCL	maximum contaminant level
mg/m <sup>3</sup>	milligrams per cubed meter
NA	not applicable
ND	non-detect
NS	not sampled
IAC	indoor air concentrations
PCBs	polychlorinated biphenyls
SRLs	Soil Remediation Levels
TCA	trichloroacetic acid
TCE	trichloroethylene
USEPA	United States Environmental Protection Agency
VOCs	volatile organic compounds
µg/L	micrograms per liter

## GLOSSARY

<i>aquifer</i>	a permeable rock stratum below the earth's surface through which groundwater moves; generally capable of producing water for a well.
<i>chemicals of concern</i>	chemicals whose concentrations are above the appropriate screening level.
<i>detection limit</i>	the minimum concentrations that must be accurately and precisely measured by the laboratory and/or specified in the quality assurance plan.
<i>dose</i>	the amount of a contamination that is absorbed or deposited in the body of an exposed organism for an increment of time. A total dose is the sum of doses received by a person from a contaminant in a given interval resulting from interaction with all environmental media that contain the contaminant. Units of dose and total dose are often converted to units of mass per volume of physiological fluid or mass of tissue.
<i>exposure</i>	an event that occurs when there is contact at a boundary between a human being and the environment with a contaminant for a specific concentration for an interval of time: the units of exposure are concentration multiplied by time.
<i>exposure pathway</i>	the process by which an individual is exposed to contaminants that originate from some source of contamination and are categorized as inhalation, dermal, and/or ingestion exposures.
<i>maximum contaminant levels</i>	enforceable drinking water standards that are protective of public health to the extent feasible.
<i>minimal risk level</i>	an estimate of daily exposure of a human being to a chemical (in mg/kg/day) that is likely to be without an appreciable risk of adverse noncancerous effects over a specified duration of exposure.
<i>parts per million</i>	a common basis of reporting water analysis. One part per million (ppm) equals 1 pound per million pounds of water.
<i>public health assessment</i>	an evaluation of relevant environmental data, health outcome data, and community concerns associated with a site where hazardous substances have been released.
<i>route of exposure</i>	means by which the contaminant actually enters or contacts the body, such

as ingestion, inhalation, dermal contact, and dermal absorption.

*soil remediation  
levels*

health-based soil screening levels. SRLs protect against toxic doses of systemic toxicants and limit excess lifetime cancer risk to one-in-one-million.

*volatile compounds*

compounds amenable to analysis by the purge and trap techniques. Used synonymously with purgable compounds.

*volatilization*

the conversion of a liquid or solid into vapors.

## **EXECUTIVE SUMMARY**

The United States Environmental Protection Agency (USEPA) asked the Arizona Department of Health Services (ADHS), Office of Environmental Health (OEH), to provide an evaluation of the health hazards that may result from exposure to soil and soil gas contaminants present on the West-Cap site, located just north of the Tucson International Airport. Improper waste disposal practices from previous industrial activities at the site have resulted in contamination of on-site soil and soil gas. The objective of this health assessment is to assess the potential adverse health impact to on-site workers from exposure to the on-site contaminated soil and soil gas. Community exposures are not presented in this public health assessment.

From the early 1960's to the early 1980's, the former West-Cap property was occupied by the now defunct and bankrupt West-Cap of Arizona Corporation which manufactured small film capacitors and magnets and used solvents as part of their manufacturing process. Solvents were discharged into floor drains that were connected to a sanitary sewer (ADEQ, 1998). Previous investigations indicate that the storage and use of solvents, such as trichloroethylene (TCE), tetrachlorethylene (PCE), 1,1-dichloroethylene (DCE) and trichloroethane (1,1,1-TCA) was conducted mainly in Buildings A, and B on the former West-Cap property. Soil gas samples taken within Buildings A and B have been found to contain low levels of TCE, DCE, PCE, and 1,1,1-TCA presenting a potential health threat to current on-site employees and visitors. Data from monitor wells suggest that groundwater underneath and downgradient of the West-Cap site has been degraded. Figures 1 shows the location of the West-Cap site..

Currently, Western Air Products Corporation (WAPC), which specializes in precision machining technologies, leases Buildings A, B, and C from the current owner Silent Sound Corporation (SSC). There are approximately 50 employees who work on the site. A quantitative analysis was conducted to determine if a public health threat exists from exposures to on-site soil and soil gas by on-site employees and visitors. On-site soil gas concentrations of TCE, DCE, PCE, and 1,1,1-TCA, within Buildings A and B located on the West-Cap site, were modeled using the Jury model (ADHS 1997a).

Soil samples did not contain any elevated levels of metals or polychlorinated biphenyls (PCBs) when compared to the current non-residential Arizona Soil Remediation Levels (SRLs). Soil gas concentrations were sampled and indoor air concentrations were modeled based on the soil gas concentrations. The results found that all indoor air concentrations were below their respective health-based ATSDR Indoor Air Comparison Values (CVs). These results show that current and future occupational inhalation exposure to contaminants found in on-site soil and soil gas present a negligible health threat for the on-site employees.

## **BACKGROUND**

### **A. SITE DESCRIPTION AND HISTORY**

The United States Environmental Protection Agency (USEPA) asked the Arizona Department of Health Services (ADHS), Office of Environmental Health (OEH), to provide an evaluation of health risks that may result from exposure to soil and soil gas contaminants present on the West-Cap site, located just north of the Tucson International Airport. Improper waste disposal practices from previous industrial activities at the site have resulted in contamination of on-site soil and soil gas. Currently, the site is operated by the Western Air Products Corporation where approximately 50 workers are employed. The objective of this health assessment is to assess if a public health hazard exists to on-site workers and visitors from exposure to the on-site contaminated soil and soil gas.

In 1981, several VOCs were detected in several Tucson Water drinking water wells near the Tucson International Airport. Subsequently, the Tucson International Airport Area Superfund Site (TIAA) was officially added to the National Priorities List (NPL) in 1983. The TIAA site consists of one main contaminated groundwater plume (commonly known as Area A) with two smaller areas of groundwater contamination, known as Western Area B and Eastern Area B, that are located to the east, and directly north of the Tucson International Airport. Western Area B is associated with the Arizona Air National Guard Center (AANG) and Eastern Area B is associated with the Burr-Brown electronics plant, and the former West-Cap facility (ADEQ 1998). Figure 1 in the Appendix shows the location of the former West-Cap facility.

The former West-Cap property is located directly north of the Tucson International Airport. From the early 1960's to the early 1980's, the former West-Cap property was occupied by the now defunct and bankrupt West-Cap of Arizona Corporation, which manufactured small film capacitors and magnets and used solvents as part of their manufacturing process. Previous investigations and interviews with facility personnel conducted by USEPA indicate that the storage and use of solvents, such as trichloroethylene (TCE), tetrachlorethane (PCE), 1,1-dichloroethylene (1,1-DCE) and trichloroethane (1,1,1-TCA) was conducted mainly in Buildings A, and B on the former West-Cap property. Waste practices included the use of a floor drain with the final discharge to a sanitary sewer (ADEQ 1998). Figure 2 in the Appendix shows the locations of the buildings on the West-Cap site.

### **B. SITE VISIT**

ADHS staff conducted a site visit on November 18 and 19, 1998 and January 20, 1999. Activities included visiting the West-Cap site and the surrounding residential areas. The following observations were made:

1. The site consists of 5 buildings located on 2207 East Elvira Rd. This northern portion of the site is empty and securely fenced off and is being bought by Burr-Brown (BB), which is located directly north of the site across the street.



2. Three of the buildings, A, B, and C which were previously inhabited by West-Cap, are currently being used by Western Air Products, a leader in manufacturing automatic precision machined parts. There are approximately 50 employees currently working in these three buildings located on the southeast portion of the site. The two other buildings are on the eastern area of the site and are subleased to two different companies.
3. Buildings A and B, known as the source areas, have several floor drains that were used by the West-Cap employees for dumping. These drains, approximately 1 foot square, are located in the cement floor, and have been filled in with dirt and covered with plastic. The drains are located in areas totally assessable to the employees.
4. A drainage channel is located just north of Buildings A, B, and C. It follows a path to the Arizona Air National Guard (AANG), located approximately one-half mile west of the West-Cap site. The AANG site is completely and securely fenced off.
5. The nearest residential area begins approximately one mile northwest of the site where several trailer parks, houses, one elementary school, and a few businesses are located.
6. The area directly north of the industrial park where the West-Cap site is located and north of Valencia Road is undeveloped and has little vegetation.

ADHS staff obtained information about the West-Cap site from current employees at the West-Cap site, Burr-Brown, the AANG, the Pima County Department of Environmental Quality (PCDEQ), and from residents who attended the community meeting on November 18, 1998 and on January 20, 1999. Additional groundwater, soil, and soil gas data were obtained from the TCE library located at the El Pueblo Clinic. The TCE library collects data and information that are provided by various governmental and community sources relating to the larger TIAA Superfund Site. This information is available to the public.

## **C. DEMOGRAPHICS, LAND USE, NATURAL RESOURCES**

### **Demographics**

The former West-Cap facility is located within the larger TIAA Superfund Site and is located directly north of the Tucson International Airport. The area surrounding the West-Cap facility includes industrial, commercial, residential, and undeveloped property. The Burr Brown property is located across the street to the north and the AANG is located approximately one-half mile west. Approximately 50 people work at this site. All employees are currently being moved from Buildings A into Building B.

The area directly north of the industrial park where West-Cap is located is undeveloped, however, there are several trailer parks and small houses located north and northwest of the AANG. Approximately, 4300 persons live in this residential area bounded by Valencia Road to the south, Nogales Highway to the west, Drexel Road to the north, and S. Campbell Ave. to the east (CDC 1999). Of these persons, approximately 1750 children under the age of 18 live in this

area.

### **Land Use and Natural Resources Use**

The West-Cap site is zoned as industrial property suitable for several types of manufacturing activities. This zoning excludes any residential usage on the site. The West-Cap facility, which closed in 1988, manufactured small film capacitors and magnetic products. These activities were primarily conducted in buildings identified as A, B, and C located on the southwest portion of the site. Currently, Western Air Products Corporation (WAPC), which specializes in precision machining technologies, leases Buildings A, B, and C from the owner Silent Sound Corporation (SSC). The northern half of the site is undeveloped and fenced off. Building D and E are located in the eastern portion of the site and are subleased to two different businesses. The WAPC is hooked up to the Tucson Water municipal water system. A majority of the residents who live near the West-Cap site obtain their drinking and irrigation water from the Tucson Water municipal water system. However, there are several private drinking and irrigation wells located throughout the residential area near the WAPC.

### **Geology**

The upper 200 feet of sedimentary material within the area show a general coarsening trend from east to west (sections D-D' & E-E'). East of the Nogales Highway where the West-Cap site is located, predominately fine-grained material is interbedded with layers and lenses of sand and gravel. The sand and gravel appear to occur in east-west trending paleochannels that represent former tributaries of the prehistoric Santa Cruz River. The climate of Tucson, Arizona is semi-arid with an average of between 10 and 11 inches of rainfall annually. Annual evaporation is about four times greater than the average annual precipitation (ADHS 1996).

### **D. HEALTH OUTCOME DATA**

ADHS staff met with both the Vice President and Maintenance Supervisor of the Western Air Products stated that no adverse health concerns have been noticed or voiced by employees. This was confirmed in brief conversations with a few of the employees who work there.

## **OCCUPATIONAL HEALTH CONCERNS**

In order to determine community health concerns, the ADHS conducted several activities to assess the health concerns of the employees at the West-Cap site and the nearby residential community. ADHS staff attended 2 community meetings on November 18, 1998, and January 20, 1999 spoke with several community residents at the meetings and in the residential areas near the West-Cap site, spoke with employees of the El Pueblo Clinic, the TCE library, the West-Cap site, and with PCDEQ personnel.

According to the Vice President, Maintenance Supervisor, and other employees at Western Air Products, no employees currently working at the former West-Cap site are concerned about being exposed to the soil contamination below Buildings A and B. The drains were very small and

don't seem to pose an idea of a health threat to employees working in Building A or B. Building A is going to be torn down in the Summer 1999 so employees are being moved to Building B.

## **ENVIRONMENTAL CONTAMINATION AND OTHER HAZARDS**

This public health assessment determines if a public health threat exists from human exposure to site-specific contaminated soil, and soil vapor. This section identifies the chemicals of concern (COC) that are related to previous activities conducted at the former West-Cap property when it was in operation. The discussion addresses the available data; the methodology used in the identification process; the criteria for selection; and determination of the extent and levels of contamination. ADHS selected the contaminants based upon the following factors:

Media that are analyzed for this health assessment include soil and soil gas. Contaminants in these media were eliminated as COCs in these media if there were no positive detections in the respective data set; or if the highest detected value was less than the corresponding screening value for each media. Screening values used in this health assessment include Soil Remediation Levels (SRLs) for soil, and health-based ATSDR Indoor Air Comparison Values (CVs) for modeled soil vapor concentrations.

Site-specific soil contaminant concentrations are compared to Soil Remediation Levels (SRLs) to determine which soil contaminants will be evaluated further in the public health assessment. The SRLs for soil ingestion were calculated by the Arizona Department of Health Services, Office of Environmental Health. SRLs are protective of human health, including sensitive groups, over a lifetime. Residential SRLs are more conservative than non-residential SRLs. Chemical concentrations in soils that exceed SRLs may not necessarily represent a health risk. Rather, when contaminant concentrations in soil exceed these standards, further evaluation may be necessary to determine whether the site poses an unacceptable risk to human health (ADHS 1997b).

Soil vapor concentrations were modeled using the Jury Model to determine corresponding indoor air concentrations (ADHS 1997a). The major quantitative modeling efforts in this health assessment determine the amount of VOCs released into the indoor air from the soil. The assumptions used are designed to produce conservative estimates of indoor air contaminant concentrations. The modeled indoor air concentrations are then compared to the health-based ATSDR Indoor Air Comparison Values (CVs) to determine if the indoor air concentrations are elevated. The selected list of soil, and soil gas contaminants indicates which contaminants will be evaluated further in the health assessment. It does not mean that the listed contaminant will cause adverse health effects from exposures.

### **A. ON-SITE CONTAMINATION**

Both soil samples and soil gas samples have been taken at the West-Cap site.

#### **Soil Sampling Results:**

In February 1996, during the Phase I Remedial Investigation, a total of eight 6 inch depth surface

soil samples including 2 duplicates were collected from beneath Buildings A and B by CH2MHILL. Six samples were analyzed for CLP Total metals and 2 samples were analyzed for PCBs using standard CLP methodology. These soil sample results were also compared to the Arizona Non-Residential SRLs. None of the constituents for which analyses were conducted were above non-residential Arizona SRLs or the residential Arizona SRLs (CH2MHILL 1996). Soil samples are provided in the Appendix in Tables A1 and A2 in Appendix A.

#### *Burr Brown Soil Sampling Results(1997)*

Burr Brown, located across the street to the north from West-Cap, is in the process of buying the northern portion of the West-Cap site, covering it with asphalt and turning it into a parking lot. This area is presently fenced off. With this in mind, Burr Brown had soil samples taken in the drainage channel which is located just north of Building A and B, and a few other locations on the empty lot. Six sediment samples, and eight soil samples of four to six feet bgs were taken on the site. The Burr Brown soil sample locations are provided in Figure 3 in Appendix A.

No volatile organic compounds (VOCs), semi-volatile organic compounds (SVOCs), total cyanide, or polychlorinated bi-phynols (PCBs) were detected in any of the samples. Eight metals were detected and all concentrations were below the Arizona Occupational SRLs with the exception of arsenic. Arsenic was detected in one soil sample at 13 mg/kg which exceeds the Arizona SRL of 10 mg/kg. Arsenic is ubiquitous in natural soils in Arizona and attributable to naturally occurring background levels. In addition, background documents do not indicate the use of arsenic-containing materials in operations performed at the site (West.Tech. 1998).

#### **Soil Vapor Sampling Results:**

Soil samples were conducted at the West-Cap site in 1996 and 1997. The 1996 samples were taken using drill rods at various locations throughout the site. Based upon this data, two soil vapor/monitoring wells were drilled in August 1997, in Buildings A and B. Subsequent soil gas sampling data was taken in the fall of 1997. Soil vapor data for 1996 and 1997 are provided in Tables A3 and A4 in Appendix A.

#### *1996 Results:*

In February 1996, during the Phase I Remedial Investigation, a shallow soil-vapor survey was conducted by CH2MHILL at the former West-Cap facility. A total of 62 samples were analyzed from 31 locations. These included 43 samples ranging from 2 to 30 feet below ground surface (bgs) and 19 quality assurance/quality control samples, utilizing USEPA Method 8010/8020, modified. This soil gas survey identified two potential source areas (Hydro Geo. 1996). Soil, and soil gas sampling locations for 1996 and corresponding contaminant levels are shown in Figures A4-7 in the Appendix A. The source areas were as follows:

1. The floor drains inside Building A where elevated levels of both TCE and PCE were detected. The highest concentration of TCE (46 µg/L) was found near the center drain at sampling location G-26, 3.5 feet below ground surface (bgs) in Building A. TCE concentrations increased toward sampling location G-32, just west of Building A. The

highest concentration of PCE was 5.6 micrograms per liter (µg/L) and was found at sampling location G-1, 5 feet bgs, near the northern drain.

2. An area near the southeast corner of Building B, where concentrations of 1,1,1 TCA and 1,1-DCE were detected. The highest concentration of 1,1-DCE of 22 µg/L, was detected at sampling locations G-10, 15 feet bgs, and G-27, 30 feet bgs. Concentrations of 1,1-DCE increased between the 6 and 15 foot depths at locations G-10, G-11, G-12, G-27, G-28, and G-33. The highest concentrations of 1,1,1-TCA of 4.3 µg/L were found at 15 feet bgs at locations G-10, 15.

The highest soil vapor concentrations of TCE, PCE, 1,1,1-TCA and 1,1-DCE were modeled using the Jury Model (ADHS 1997a) to determine corresponding indoor air concentrations. As shown in Table 1 below, the soil vapor mean concentrations were below the corresponding ATSDR Indoor Air Comparison Values.

**Table 1: Contaminant Concentrations of 1996 On-Site Soil Gas Samples, Respective Modeled Indoor Air Concentrations and Comparison to ATSDR's\* Air Comparison Values.**

Contaminant	Concentration Range in Soil Vapor (µg/L) <sup>†</sup>	Mean Concentration in Soil Vapor (µg/L)	Modeled Indoor Air Concentration (µg/L)	ATSDR Air CV <sup>‡</sup> (µg/L)	Exceed ATSDR CV?
TCE <sup>§</sup>	ND <sup>¶</sup> - 46	3.58	2.0E-07	6.0E-04	NO
PCE <sup>**</sup>	ND - 5.6	0.5	4.4E-08	2.0E-03	NO
1,1-DCE <sup>††</sup>	ND - 22	3.59	1.8E-08	2.0E-05	NO
1,1,1-TCA <sup>‡‡</sup>	ND - 4.3	53.62	3.0E-06	6.0E-04	NO

\* ATSDR = Agency for Toxic Substances and Disease Registry; <sup>†</sup> ug/L = micrograms per liter; <sup>‡</sup> CV = Comparison Values;

<sup>§</sup> TCE = trichloroethylene; <sup>¶</sup> ND = non-detect; <sup>\*\*</sup> PCE = tetrachloroethane; <sup>††</sup> 1,1-DCE = 1,1-dichloroethylene; <sup>‡‡</sup> 1,1,1-TCA = 1,1,1 trichloroethane. CV for TCE is also used for TCA

### *Soil Vapor Results (1997)*

In 1997, two soil vapor/groundwater monitor wells, WC1 and WC2, were drilled in Buildings A and B, respectively, to a depth of 121 feet bgs. Locations of WC1 and WC2 are provided in Figure 8. Two rounds of soil gas samples was taken by CH2MHILL in September, and November. The highest concentrations of TCE and PCE were in Well WC-1 in Building A at 111 µg/L (with the exception of a duplicate concentration of 133 µg/L) and 28.7 µg/L, respectively. The highest concentrations of DCE of 140 µg/L and TCA of 3.28 µg/L were in Well WC-2 in Building B. As shown in Tables 2 and 3 below, the highest on-site soil vapor concentrations were below the corresponding ATSDR Indoor Air Comparison Values. Soil gas results from 1997 are presented in Tables 2 and 3 below. The 1997 soil samples are shown in Table A1 in Appendix A.

**Table 2: Contaminant Concentrations of 1997 On-Site Soil Gas Samples, Respective Modeled Indoor Air Concentrations and Their Comparison to ATSDR\* Indoor Air Comparison Values for Well WC-1.**

Contaminant	Concentration Range in Soil Vapor (µg/L) <sup>†</sup>	Mean Concentration in Soil Vapor (µg/L)	Modeled Indoor Air Concentration (µg/L)	ATSDR Air Comparison Value (µg/L)	Exceed ATSDR CV <sup>‡</sup> ?
TCE <sup>§</sup>	ND <sup>¶</sup> - 111.0	89.62	5.2E-06	6.0E-04	NO
PCE <sup>**</sup>	ND - 28.7	16.87	1E-06	2.0E-03	NO
1,1-DCE <sup>††</sup>	ND - 1.24	0.56	3.0E-09	2.0E-05	NO

\* ATSDR = Agency for Toxic Substances and Disease Registry; <sup>†</sup> µg/L = micrograms per liter. All chemicals that were detected in less than 5% of the soil sample data were not included in the data set; <sup>‡</sup> CV = Comparison Values; <sup>§</sup> TCE = trichloroethylene; <sup>¶</sup> ND = non-detect; \*\* PCE = tetrachloroethane; <sup>††</sup> 1,1-DCE = 1,1-dichloroethylene;

**Table 3: Contaminant Concentrations of 1997 On-Site Soil Gas Samples, Respective Modeled Indoor Air Concentrations and Their Comparison to ATSDR\* Indoor Air Comparison Values for Well WC - 2.**

Contaminant	Concentration Range in Soil Vapor (µg/L) <sup>†</sup>	Mean Concentration in Soil Vapor (µg/L)	Modeled Indoor Air Concentration (µg/L)	ATSDR Air CV <sup>‡</sup>	Exceed ATSDR CV?
TCE <sup>§</sup>	ND <sup>¶</sup> - 1.27	0.74	4.40E-08	6.0E-04	NO
PCE <sup>**</sup>	ND - 3.48	1.2	9.5E-08	2.0E-03	NO
1,1-DCE <sup>††</sup>	ND - 140	68.42	3.5E-07	2.0E-05	NO
1,1,1-TCA <sup>‡‡</sup>	ND - 3.28	1.62	9.3E-08	6.0E-04	NO

\* ATSDR = Agency for Toxic Substances and Disease Registry; <sup>†</sup> µg/L = micrograms per liter; <sup>‡</sup> CV = Comparison Values; <sup>§</sup> TCE = trichloroethylene; <sup>¶</sup> ND = non-detect; \*\* PCE = tetrachloroethane; <sup>††</sup> 1,1-DCE = 1,1-dichloroethylene; <sup>‡‡</sup> 1,1,1-TCA = 1,1,1 trichloroethane. CV for TCE is also used for TCA.

In summary, contaminants found in soil and soil vapor data are below their corresponding standards and respective ATSDR Comparison Values.

## **B. OFF-SITE CONTAMINATION**

This health assessment does not review off-site contamination. Off-site groundwater contamination will be addressed in a separate public health assessment.

## **C. PHYSICAL AND OTHER HAZARDS**

During the first site visit, it was noted that there were approximately 20 - 30 drums that were stacked on top of each other being stored between Building A and B. This is an area where employees travel back and forth between the two buildings. There were additional drums

stacked on the eastern side of Building B but these were in an area less accessible to employee traffic. All employees had been moved into Building B by the time of the second visit on January 20, 1999. The Maintenance Supervisor of Western Air Products stated that Building A is scheduled to be torn down in May or June 1999. A fence has been put up around Building A with access to the side doors. The drums have been moved. Only wood pallets remain.

## **PATHWAY ANALYSES**

To determine whether on-site workers and visitors are currently or could be exposed to contaminants from the West-Cap site in the future, ADHS evaluated the environmental and human components that led to human exposure. This pathway analysis consists of five elements. These are as follows: a source of contamination, transport through an environmental medium, a point of exposure, a route of exposure, and an exposed population. ATSDR categorizes an exposure pathway as a completed current or potential future exposure pathway. Completed pathways require that the all five elements exist and indicate that exposure to a contaminant has occurred in the past, present, or could occur in the future. An exposure pathway is considered incomplete and can be eliminated if at least one of the five elements is missing and will never be present. Completed current and potential exposure pathways are discussed below.

### **A. COMPLETED CURRENT EXPOSURE PATHWAYS**

The two current receptor populations that have been identified include on-site workers and visitors. The complete current exposure pathways identified include possible ingestion and inhalation exposure to on-site contaminated surface soil, and inhalation exposures to contaminated soil gas by workers at the site and visitors.

#### **Soil Pathway**

Past soil samples taken near the drains in both Buildings A and B imply that soil contamination has occurred beneath these two buildings. Past, current, and future exposures may result from the contamination of these soils. While this may be the case, the soil concentrations are well below their respective SRLs and pose no current or future health threat from ingestion, inhalation, or dermal exposures by the employees working on the site or visitors.

There were eight soil samples including two duplicates, taken at 6 inches depth, within Buildings A and B. These samples may not adequately characterize additional soil contamination either at depths below 6 inches or in other areas below the foundation of Buildings A and B or in the outer perimeter. Therefore, we cannot assume that the results of the soil data are completely representative of the entire soil contamination on this site. Destruction of Building A and subsequent soil remediation is planned for the summer 1999. Western Products has moved employees from Building A into Building B. This will reduce the amount of future exposures to contaminated soil via ingestion, inhalation, or dermal exposures that otherwise could occur.

#### **Soil Vapor Pathway**

Past, current, and future exposure pathways may result from inhalation of soil gas vapors

escaping from the floor drains and cracks in the foundation of Buildings A into the working areas, and through cracks in the concrete slab outside to the east of Building B. Samples were taken in Building A where the floor drains are located, in some perimeter areas including the wash and in areas where surface water pooling may occur. It appears that the soil vapor data may be fairly representative of the entire contaminated area. Potential future exposures will be reduced since Western Air Products have moved employees out of Building A.

**Table 4: Complete Past and Current Exposure Pathways for the West-Cap Site**

Pathway Name	EXPOSURE PATHWAY ELEMENTS					Time
	Source	Environmental Media	Point of Exposure	Route of Exposure	Exposed Pop.	
Surface Soil	Buildings A	Surface Soil	Drains in buildings A.	Ingestion Inhalation Dermal	Workers	Past Current
Soil Vapor	Buildings A and B	Soil	Drains in buildings A. Cracks in the foundation of Building B and outside area.	Inhalation	Workers	Past Current

#### **A. POTENTIAL FUTURE EXPOSURE PATHWAYS**

Building A is expected to be torn down and remediation begun in summer of 1999. During the remediation process for Building A, construction workers may potentially be exposed via ingestion, inhalation, and dermal exposures to the contaminated soil and soil gas. On-site employees may potentially be exposed via inhalation to contaminated fugitive dust. However, the actual exposures cannot be determined at this time. Future plans also include extending Building B westward to the eastern edge where Building A is currently located. This will also create additional disturbance of any underlying contaminated soil.

Areas of higher concentration of 1,1-DCE and 1,1,1-TCA were found in an outside area southeast of Building B in a storage area. The storage area is covered with cement and has a metal roof. There are no walls so it is open to the elements. However, employees have access to this area and may be exposed to soil gas via inhalation.



**Table 5: Potential Future Exposure Pathways for the West-Cap Site**

Pathway Name	EXPOSURE PATHWAY ELEMENTS					Time
	Source	Environmental Media	Point of Exposure	Route of Exposure	Exposed Pop.	
Surface Soil	Building A	Surface Soil	Cracks and drains in Building A	Ingestion Inhalation Dermal	Construction Workers	Future
Soil Vapor	Buildings A and B	Soil	Drains in buildings A. Cracks in foundation of Building B and in outside storage area	Inhalation	Employees, Construction Workers	Future

## **PUBLIC HEALTH IMPLICATIONS**

This section reviews the potential for adverse health effects in on-site workers and visitors exposed to specific contaminants through completed current or potential future exposure pathways. ADHS analyzed the on-site exposure scenarios to determine what, if any public health hazard exists from exposure to contamination at the site.

### **A. TOXICOLOGICAL EVALUATION**

If contaminant levels are found to be above their respective standards, chronic daily intakes (CDIs) are then calculated and compared to the corresponding ATSDR Minimal Risk Levels (MRL). The MRL is an estimate of daily human exposure to a contaminant below which non-cancer, adverse health effects are unlikely to occur. MRLs are not used to determine the specific adverse health effects of exposure. Rather, they are used to determine if there is the need for more thorough contaminant specific investigation. MRLs are not used to determine a safe level of contaminants at a site. MRLs are developed for each route of exposure, such as inhalation and ingestion, and for a length of exposure, such as acute (less than 14 days), intermediate (14 to 356 days), and chronic (greater than 365 days). Therefore, if exposure doses are found to be above their respective MRLs further evaluation is made to assess the health risks.

Contaminant concentrations found in soil, and soil gases were below the respective non-residential and residential SRLs, and ATSDR Indoor Air Comparison Values, respectively. This indicates that the exposure doses will be far below their respective MRLs. Therefore, estimates of pathway specific exposure intake doses and exposure concentrations did not have to be

quantified for any specific on-site exposure pathways. As described previously in this assessment, chemicals were eliminated as chemicals of concern (COCs) in these media if there were no positive detections in the respective data set; or if the highest detected value was less than the corresponding standard for each media. Since all site related contaminants met these criteria, it was not necessary to calculate exposure intakes for further assessment.

Current and future inhalation exposures to the on-site soil gas, and dermal, ingestion and inhalation exposures to contaminated soil by on-site workers and visitors do not currently pose a public health hazard. The following sections describe this conclusion in more detail.

### **Soil Exposures**

There is a possibility that on-site employees and visitors are currently being exposed to soil contaminated with various metals found in Buildings A and B. However, those levels were found to be below the Arizona occupational and residential SRLs. All other soil concentrations for metals and PCBs in Buildings A and B do not exceed the respective non-residential or residential respective SRLs. Therefore, chronic daily exposure to the current soil concentrations identified are unlikely to pose a health hazard to employees working on the site or visitors. Future exposures to contaminated soil in Building A will not continue much longer since employees have been moved to Building B.

### **On-site Soil Gas Exposures**

There is a possibility that on-site employees are currently being exposed to soil vapors contaminated with TCE, PCE, 1,1-DCE, and 1,1,1-TCA. The soil vapors were modeled using the appropriate Jury Model to determine the corresponding indoor air concentrations (ADHS 1997c). The Jury Model is a conservative model which assumes there is no clean soil layer above the contamination. Results of this modeling suggest that the estimated indoor air concentrations of these contaminants in Buildings A and B are below respective health-based ATSDR's Air Comparison Values (CVs). Therefore, chronic daily exposure to the current soil gas concentrations identified are unlikely to pose a health hazard to employees working on the site. The highest concentrations of TCE and PCE were found in Well WC-1 just outside building A. Exposures to these levels of TCE and PCE will not continue much longer in Building A since employees are currently being moved into Building B. The highest concentrations of 1,1-DCE and 1,1,1-TCA were found in Well WC-2 just outside Building B.

## **B. ATSDR'S CHILD HEALTH INITIATIVE**

The ADHS has prepared this public health assessment under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). ADHS has included the following information in accordance with ATSDR's Child Health Initiative.

Sub-populations of concern are sensitive receptor populations who may be particularly susceptible to chemical exposure. They may include infants, the elderly, or individuals with respiratory problems depending on the COC's and the nature of the exposures. Often exposure points for sensitive receptors include hospitals, nursing homes, schools, and day care centers

where these populations gather. None of these facilities are present in the industrial area on or near the West-Cap site. Any sensitive persons who visit the Western Air Products would be on-site for very short periods of time limiting any chronic exposure to the contamination. This has been limited even more since all business activities are being moved to Building B. The concentrations of contaminants detected in soil and soil gas are not high enough to produce acute adverse health effects.

ATSDR's Child Health Initiative recognizes that the unique vulnerabilities of infants and children demand special emphasis in communities faced with contamination of their water, soil, air, or food. Children are at greater risk than adults from certain kinds of exposures to hazardous substances emitted from waste sites and emergency events. They are more likely to be exposed because they play outdoors and they often bring food into contaminated areas. They are shorter than adults, which means they breathe dust, soil, and heavy vapors close to the ground. Children are also smaller, resulting in higher doses of chemical exposure per body weight. The developing body systems of children can sustain permanent damage if toxic exposures occur during critical growth stages. Most important, children depend completely on adults for risk identification and management decisions, housing decisions, and access to medical care.

## **CONCLUSIONS**

This public health assessment evaluates the public health hazards associated with exposures to human receptors under current and potential future land-use conditions to chemicals in soil at the former West-Cap property. ADHS concludes that no human health hazard exists for visitors and employees at the former West-Cap site

Soil samples did not contain elevated concentrations of metals or PCBs when compared to the respective residential SRLs. Soil gas concentrations of TCE, DCE, PCE, and 1,1,1 within Buildings A and B located on the West-Cap site were modeled and found to be below their respective health-based ATSDR Indoor Air Comparison Values. Therefore, current and future occupational inhalation exposures to contaminants detected in on-site soil gas do not pose a health hazard to employees working on-site.

In summary, ADHS concludes that no apparent public health hazard exists as a result of ingestion, dermal, or inhalation exposures by employees and visitors to the on-site soil and soil gas contamination on the West-Cap site.

## **PUBLIC HEALTH ACTION PLAN**

The Public Health Action Plan (PHAP) for the West Cap Site contains a description of actions taken, to be taken, or under consideration by ATSDR and ADHS at and near the site. The purpose of the PHAP is to ensure that this public health assessment not only identifies public health hazards, but also provides a plan of action designed to mitigate and prevent adverse

human health effects resulting from exposure to hazardous substances in the environment. ADHS and ATSDR will follow up on this plan to ensure that actions are carried out.

#### *Actions Completed*

1. ADHS attended a public meeting at the Tucson UCAB (Unified Community Action Board) in November 1998, January and February 1999 to meet with staff from USEPA and other regulatory agencies about the public health assessment for the West Cap Site.
2. In February 1999, ADHS met with the management at the West Cap site to discuss the health assessment. The site visit included taking pictures, identifying the drains, researching data at the TCE Library, and talking with residents in the area to identify their concerns.
3. In February 1999, ADHS met with USEPA in San Francisco to discuss the health assessment and risk assessment for the West Cap site.
4. In April 1999, ADHS met with USEPA, PCDEQ, ADEQ, and other parties in San Francisco for 2 days to discuss the West Cap and Plume B situation in Tucson. Plans were put in place to better characterize the site, determine the source of the contamination, and further the clean up of West Cap.
5. In March 1999, ADHS submitted a final draft of the West Cap Site public health assessment and risk assessment to ATSDR.
6. In September, ADHS staff attended the UCAB meeting and presented the results of the health assessment and other health assessments being conducted on the Tucson site.

#### *Actions Proposed*

1. ADHS will continue to meet with the community residents at the UCAB meetings on a regular basis to communicate the ADHS activities being conducted at the Tucson site. Specific goals are to increase the understanding of the technical aspects of the area contaminants and their fate and transport, and to educate the community on ways to minimize their exposures to site-related contaminants and physical hazards.
2. ADHS will continue to work with USEPA, ADEQ, PCDEQ, and other consulting companies to address additional health concerns about exposures at the West Cap Site.

### **RECOMMENDATIONS**

Although this assessment has determined that no current health threat exists to on-site employees the ADHS recommends the following:

1. As a precaution, when Building A is torn down, construction workers and other

employees that have access to the area need to be advised on adequate protection measures to be used during the demolition process in order not to be exposed to on-site contaminated soil and soil gas.

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## APPENDIX A

### ◆Tables

Table A1	Soil Sampling Results for Metals (1996)
Table A2	Soil Sampling Results for polychlorinated biphenyls (PCBs) (1996)
Table A3	Summary of Soil Gas Sampling Results (1996)
Table A4	Soil Vapor Sampling Data Summary (1997)
Table A5	Calculations for Indoor Air Concentrations Using the Jury Model

### ◆Figures

Figure 1	Location of West-Cap site and Tucson International Airport
Figure 2	Location of Buildings on West-Cap Site.
Figure 3	Burr Brown Soil Sample Locations in Wash and Vacant Lot
Figures 4-7	Locations of 1996 Soil and Soil Gas Samples in Buildings A and B
Figure 8	Location of Wells WC1 and WC 2 and 1997 Soil Vapor Samples

### ◆Jury Model

### ◆ADHS Toxicological Profiles for 1,1-DCE, PCE, TCE, 1,1-TCA

## Tables and Figures

## Indoor Air Concentrations: Jury Model

Soil gas exposures were evaluated using the Juror Indoor Air Model to estimate indoor air exposure concentrations. The model assumes that gas entering a structure is instantaneously mixed within the entire volume. VOC concentrations within a building are a function of the flux from the soil, the area and volume of the building, and the rate of air exchange for the structure. This is a common assumption for assessments of this type and is intended as an upper bound estimate. It should be recognized that anytime a model is used the uncertainty of the estimated quantities is greater than if an accurate measurement were taken. These estimates assume that each VOC detected in soil gas is uniformly present beneath each building at the maximum concentration detected during the investigation. Therefore, this screening level analysis very likely overestimates concentrations of soil gas contaminants in indoor air.

Data used for the soil gas exposure assessment included all results previously presented in this health assessment. Indoor air concentrations were estimated using the following model:

### Jury Model

Jury et al. have developed an equation for estimating the flux of a contaminant from a finite source of contaminated soil (Jury et al. 1990). The model is applicable when contamination extends from the surface for a known fixed thickness in the soil column. The model is applicable when there is no clean soil layer above the contamination. The following assumptions were used to derive this simplified equation:

- Uniform soil properties (homogeneity)
- Instantaneous linear equilibrium adsorption
- Linear equilibrium liquid-vapor partitioning
- Uniform initial contaminant concentration
- Contaminants present at less than saturation
- No clean soil layer
- No water evaporation or leaching
- No biological or chemical degradation

$$J_s = C_o \left( \frac{D_A}{\pi \tau} \right)^{1/2} \left[ 1 - \exp \left( \frac{-d_s^2}{4D_A\tau} \right) \right]$$

Equation - E

where:

$J_s$	=	contaminant flux at ground surface (g/cm <sup>2</sup> -s)
$C_o$	=	uniform contaminant concentration at $\tau = 0$ (g/cm <sup>3</sup> )
$D_A$	=	apparent diffusivity (cm <sup>2</sup> /s)

$\pi$	=	3.14
$\tau$	=	time(s)
$d_s$	=	depth from the soil surface to the bottom of contamination at $\tau=0$ (cm),

and where:

$$D_A = \frac{\left[ \left( \frac{\Theta_a^{10/3} D_i H' + \Theta_w^{10/3} D_w}{n^2} \right) \right]}{(\rho_b K_d + \Theta_w + \Theta_a H')}$$

Equation - F

where:

$\Theta_a$	=	air-filled soil porosity ( $L_{air}/L_{soil}$ ) = $n - \Theta_w$
$n$	=	total soil porosity ( $L_{pore}/L_{soil}$ ) = $1 - (\rho_b/\rho_s)$
$\Theta_w$	=	water-filled soil porosity ( $L_{water}/L_{soil}$ ) = $w \rho_b/\rho_w$
$\rho_b$	=	soil dry bulk density (g/cm <sup>3</sup> )
$\rho_s$	=	soil particle density (g/cm <sup>3</sup> )
$w$	=	average soil moisture content (g/g)
$\rho_w$	=	water density (g/cm <sup>3</sup> )
$D_i$	=	diffusivity in air (cm <sup>2</sup> /s)
$H'$	=	dimensionless Henry's law constant = $41 \times H$
$H$	=	Henry's law constant (atm-m <sup>3</sup> /mol)
$D_w$	=	diffusivity in water (cm <sup>2</sup> /s)
$K_d$	=	soil-water partition coefficient (cm <sup>3</sup> /g) = $K_{oc} f_{oc}$
$K_{oc}$	=	soil organic carbon partition coefficient (cm <sup>3</sup> /g)
$f_{oc}$	=	organic carbon content of soil (g/g)

To estimate the average contaminant flux over 30 years, the time-dependent contaminant flux must be solved for various times and the results averaged. A simple computer program or spreadsheet can be used to calculate the instantaneous flux of contaminants at set intervals and numerically integrate the results to estimate the average contaminant flux. The time-step interval must be small enough (e.g., 1-day intervals) to ensure that the cumulative loss through volatilization is less than the total initial mass. Inadequate time steps can lead to mass-balance violations (USEPA 1996a).

Alternatively, the EMSOFT computer program developed by the USEPA Office of Research and Development National Center for Environmental Assessment (NCEA) may be used to estimate average flux. The computer program provides an average emission flux over time by using an analytical solution to the integral, thereby eliminating the problem of establishing adequate time

steps for numerical integration. EMSOFT is available through the NCEA in Washington, D.C.

## **Toxicological Profiles**

Below are toxicological profiles summaries for 1,1-DCE, PCE, TCE, and 1,1,-TCA. However, it should be noted that a number of assumptions are utilized in computing human exposure and toxicity for individual chemicals. These assumptions combine conservative exposure estimates that are still within the range of possible exposures with toxicity data that assume that a maximal effect can be expected in humans following exposure to any chemical.

For example, there are several sources of uncertainty that affect the characterization of health affects from chemicals through various exposure pathways. Much of the exposure estimates are based on dose-response relationships observed, primarily, in experimental animals. There may be differences between animals and humans in metabolic response to a chemical. The test animals may have genetic predispositions that are not considered. Oftentimes, high doses are administered to small populations of animals and then low dose response is estimated by extrapolation. Experimental animals have naturally short life spans, whereas humans do not. The toxicity values used were developed singly and responses may differ when complex mixtures are present.

Another issue is whether the animal concentration of a chemical per unit of body weight ( $\text{mg}/\text{m}^2$  or  $\text{mg}/\text{kg}$ ) will result in the same level of toxicity for a human measured in like units. This assumes that absorption of a chemical (toxicokinetics) as seen in animals will be similar in humans and, therefore, similar health effect will result. In reality, this may not be the case.

It is also assumed that any concentration of a chemical greater than zero has a probability of producing a carcinogenic response. Because of the body's ability to clear foreign substances, this may not be true. The exact concentration for specific chemicals at which the body mechanisms fail and cancer growth occurs is unknown.

In addition, it is assumed that any animal carcinogen is also a human carcinogen. This assumption is made even without research into human effects for the particular substance. Because of limited human data, information for slope factors is gained from animal studies. Since animals (rodents) differ somewhat anatomically (e.g. no sweat glands in rats) and physiologically (e.g. less efficient DNA repair in rats) from humans, the mechanism of cancer induction may differ markedly between animals and humans.

### **1,1-Dichloroethylene (DCE)**

ATSDR has derived an MRL of 0.02ppm for intermediate-duration inhalation exposure based on a NOAEL of 5 ppm for liver effects in guinea pigs. The study used to derive the MRL was conducted by Quast et al.1983. The mean indoor air concentration of 1,1-DCE at the West-Cap site was well below the respective ATSDR health based Indoor Air Comparison Value which indicates it is below the MRL. Therefore, no adverse health effects would be expected from exposures to these amounts of 1,1-DCE in the soil gas based upon the exposure scenario described earlier in this document.

1,1-Dichloroethylene (CAS No. 75-35-4,  $C_2H_2Cl_2$ ) is a halogenated hydrocarbon made by man. Synonyms include 1,1-dichloroethene; 1,1-DCE; and vinylidene chloride. DCE is used to manufacture packing wrap (Saran™) and flame-retardant fabrics. DCE is released primarily into air and water from industrial emissions, hazardous waste sites, and accidental spills. The highest potential exposure levels are seen in occupations utilizing DCE and in populations residing near hazardous waste sites.

The routes of exposure for DCE include inhalation, ingestion, or dermal contact. No human studies were available for the absorption, distribution, metabolism, and excretion of DCE. In animal studies, DCE was readily absorbed following inhalation and ingestion exposures and was distributed to the kidneys, liver, and lungs on inhalation and to the kidneys and liver on ingestion. The metabolic pathway of DCE in rats has been extensively studied with formation in the initial stages of an epoxide intermediate. With inhalation exposure, the majority of the DCE metabolites was excreted in the urine with very little eliminated unchanged in the expired air. In an ingestion study of rats, the greatest portion of the DCE was excreted in the urine (44-80%) and recovered as  $CO_2$  (5-14%) with 1% unchanged in expired air and a small amount in the feces.

Upper airway irritation, a high incidence of liver toxicity in workers of a DCE polymerization plant, and CNS depression (convulsions, spasms, unconsciousness) have been demonstrated in humans with inhaled DCE. In addition, animal research has demonstrated that DCE is a weak teratogen and also causes reproductive effects and DNA damage with inhalation. Toxic effects in humans were not available for ingestion exposure. However, oral animal studies produced adverse outcomes to the gastrointestinal (forestomach edema) and respiratory (pulmonary edema) systems, to the liver (necrosis, hemorrhage), and to fetal development (increase in mean fetal crown-rump length in pups). With human dermal exposure, local irritant effects were observed.

Three human studies investigated the association of inhalation exposure to DCE and the development of cancer. No association was discovered, but the studies had real limitations such as small sample sizes. Animal studies have reported an increase in kidney and mammary cancers and lung tumors with inhalation exposures. Liver cancer was seen in oral animal studies. Dermal application of DCE in mice demonstrated its tumor initiator effect. DCE has an USEPA Weight-of-Evidence Classification of C (possible human carcinogen).

### **Tetrachloroethylene (PCE)**

ATSDR has derived a chronic duration MRL of .04 ppm with an uncertainty factor of 100. It was derived based on increased reaction times in workers exposed to PCE in dry cleaning shops at an average concentration of 15 ppm for about 10 years. (Ferroni et al. 1992). The mean indoor air concentration of PCE at the West-Cap site was well below the ATSDR health based Indoor Air Comparison Value which indicates it is below the MRL. Therefore, no adverse health effects would be expected from exposures to the soil gas concentrations of PCE based upon the exposure scenario described earlier in this document.

Tetrachloroethylene (CAS No. 127-18-4,  $C_2Cl_4$ ) is a halogenated hydrocarbon which is man



made. Synonyms include carbon bichloride; carbon dichloride; ethylene tetrachloride; perchloroethylene; tetrachloroethene; and 1,1,2,2-tetrachloroethylene. PCE is commonly used as an industrial solvent and degreaser, as an intermediate for manufacturing other chemicals, and is used extensively in the dry cleaning and textile industries. Although PCE is liquid at room temperature, it tends to evaporate into the atmosphere which accounts for most of its environmental emissions, especially from the industrial and dry-cleaning operations. Exposure to PCE results from employment in certain industries (e.g. dry cleaning), residence near emission sites, and ingestion of contaminated food and water.

The routes of exposure for PCE include inhalation, ingestion, or dermal contact. Absorption following inhalation or ingestion is extensive but poor with dermal exposure. Following absorption, much of the inhaled and ingested PCE is deposited in the fatty tissue. PCE was reported to be distributed in the liver, kidney, brain, and lung of a dry cleaner who received a fatal inhalation exposure to PCE.

Inhalation exposure to PCE has affects a number of body systems. Respiratory irritation was documented in volunteers exposed to a concentration of 216 ppm for 45 minutes to 2 hours workers and in workers exposed to PCE at inhalation levels of 232-385 ppm.

The liver has been a target organ in PCE exposure. Hepatocellular damage was diagnosed in a woman worker inhaling PCE fumes. In addition, a dry cleaner, who was exposed to PCE fumes, was found to have diffuse fatty liver and died shortly after the exposure. This condition may have existed prior to employment. No effect to the liver (as measured by the presence of liver enzymes - alanine aminotransferase) was observed in 22 dry cleaning workers who received a TWA exposure to PCE of 21 ppm.

Renal effects have additionally been observed based upon the inhalation dose. An examination of the workers with an estimated TWA of 10 ppm of PCE for 14 years demonstrated a rise in urinary levels of lysozyme and  $\beta$ -glucuronidase indicating mild tubular damage of the kidney. Serum creatinine and urinary albumin,  $\beta$ - $\mu$ -globulin and retinol-binding protein levels were found to be within normal limits in dry cleaning workers exposed to a TWA of 21 ppm of PCE for six years.

Data to evaluate immunological effects from inhalation exposure are not firm. However, in one study, mice were found to be more sensitive to pulmonary bacterial infection following a three hour inhalation exposure to PCE at 50 ppm.

Neurological effects have additionally been observed in humans. The brain is the target organ for exposure by inhalation. Impaired perceptual and intellectual function and attention were detected in dry cleaning workers exposed to a TWA of 12 ppm (for 141 days) or 54 ppm (for 127 days) of PCE when compared to controls. In a separate study of dry cleaning workers, no significant changes in neurological symptoms or psychomotor performance were seen in the workers who received a TWA exposure to PCE of 21 ppm over an average of six years. However, in 17 of 22 subjects, neurologic symptoms (memory loss and difficulty sleeping) were more widespread in the exposed compared to the control group. In one study, headache, dizziness, difficulty speaking, and sleepiness occurred after inhalation exposure to 100 ppm of

PCE for seven hours. After a one hour exposure to 106 ppm, volunteers of one exposure study manifested no symptoms of neurological impairment. At 216 ppm for an exposure period of 45 minutes to 2 hours, symptoms of dizziness and drowsiness developed.

PCE has an USEPA Weight-of-Evidence Classification of B2 (probable human carcinogen). The USEPA is presently reviewing PCE's Weight-of-Evidence classification and slope factor. Pending USEPA's final report, this study utilizes the existing information on classification and slope factor.

### **Trichloroethylene (TCE)**

ATSDR has derived an intermediate duration inhalation MRL of 0.1 ppm with an uncertainty factor of 300 based on neurological effects in rats (Arito et al. 1994a). The mean indoor air concentration of TCE at the West-Cap site was well below the ATSDR health based Indoor Air Comparison Value which indicates it is below the MRL. Therefore, no adverse health effects would be expected from exposures to the soil gas concentrations of TCE based upon the exposure scenario described earlier in this document.

Trichloroethylene (CAS No. 79-01-6,  $C_2HCl_3$ ) is a halogenated hydrocarbon. Synonyms include 1-chloro-2,2-dichloroethylene; 1,1-dichloro-2-chloroethylene; ethylene trichloride; and 1,1,2 trichloroethylene. TCE is used as an industrial solvent and degreaser, an intermediate for manufacturing other chemicals, and is commonly used in the automotive, metal, and textile industries. In the past, it has also been used as a general and obstetrical anesthetic, surgical disinfectant, and extractant of caffeine for decaffeinated coffee. Although TCE is liquid at a room temperature, evaporation does occur in industrial processes resulting in exposure by inhalation for workers and the general public residing in areas of industry and waste disposal sites. The degreasing operation in industry is the primary cause of TCE emissions into the environment with releases also occurring from other industries and disposal of waste.

Routes of exposure for TCE include inhalation, ingestion, or dermal contact. Human absorption following inhalation or ingestion is extensive, but poor with dermal exposure.

Inhalation exposure has also resulted in health effects. Neurological outcomes have been observed in humans following exposure by inhalation. Concentrations of 0, 27, 81, or 201 ppm of TCE were administered to groups of three human subjects for four hours. Irritation of the eyes and throat and drowsiness were observed at 27 ppm or greater, headache at 81 ppm or greater, and dizziness and anorexia at 201 ppm. In another study, subjects were found to have a significant reduction in performance on a perception test, the Wechsler Memory Scale, a complex reaction time test, and manual dexterity tests with inhalation exposure of 110 ppm of TCE for two four hour exposures with an interval of 1.5 hours. In contrast, no significant treatment-related effects on behavioral task performance were reported in subjects receiving 95 ppm or 150 ppm or 300 ppm of exposure to TCE for 2.5 hours. Subjects were tested for reaction time, hand steadiness, hand tapping, and pursuit tracking. In a separate study, however, eight male human volunteers were found to have a compromise in visual-motor skills. Subjects were

exposed to 0, 100, 300, or 1,000 ppm of TCE for two hour periods with an interval of three days between exposure sessions. A significant reduction in visual-motor performance was seen at an exposure of 1,000 ppm. Subjective symptom, neurological, and psychiatric evaluations have been done in yet another study.

Workers who had been employed an average of 3.75 years in industrial operations utilizing TCE as a solvent were found to have a greater likelihood of complaints which included vertigo, fatigue, and headaches with test results showing short-term memory loss, fewer word associations, and increase misunderstanding at higher mean concentrations of TCE (85 ppm). The high-dose workers were compared to those exposed to lower mean concentrations of TCE (14 or 34 ppm).

Severe liver damage in the form of necrosis has been observed in acute occupational inhalation exposure to lethal concentrations of TCE. Kidney dysfunction and failure have also been seen with inhalation exposures in acute occupational and intentional exposure. Immune system effects as manifested by the change in weight of the thymus gland have been observed in rats receiving continuous exposure at 800 ppm of TCE. Hematological effects in the form of depression of delta-aminolevulinate dehydratase activity was reported in rats with continuous inhalation exposure for 10 days. The reduced activity was detected in liver and bone marrow cells at 50 ppm or greater and in erythrocytes at 398 ppm or greater.

### **1,1,1-Trichloroethane**

ATSDR has derived an intermediate duration inhalation MRL of 0.7 ppm based on a study by Rosengren et al. (1985). This study found evidence of astrogliosis (increased glial fibrillary acid protein levels) in the brains of gerbils exposed to 210 or 1,000 ppm, but not 70 ppm of 1,1,1-TCA continuously for 3 months. OSHA requires employers of workers who are occupationally exposed to 1,1,1-TCA to institute engineering controls to maintain employee exposures at or below permissible exposure limits (PEL) of 350 ppm. The mean indoor air concentration of 1,1,1-TCA at the West-Cap site was is well below the ATSDR health based Indoor Air Comparison Value which indicates it is below the MRL. Therefore, no adverse health effects would be expected from exposures to the soil gas concentrations of 1,1,1-TCA based upon the exposure scenario described earlier in this document.

1,1,1-TCA is used as a solvent for adhesives (including food packaging adhesives) and in metal degreasing, pesticides, textile processing, cutting fluids, aerosols, lubricants, cutting oil formulations, drain cleaners, shoe polishes, spot cleaners, printing inks, and stain repellents, among other uses. It is used in industry primarily for cold-cleaning, dip cleaning, bucket cleaning, and vapor degreasing operations of items such as precision instruments, molds, electrical equipment, motors, electronic components and instruments, missile hardware, paint masks, photographic film, printed circuit boards, generators, switchgears, semiconductors, and

high vacuum equipment, fabrics, and wigs. It is also used for on-site cleaning of printing presses, food packaging machinery, and molds. 1,1,1-TCA was formerly used as a food and grain fumigant (ATSDR, 1994).

Upon first exposure, 1,1,1-TCA rapidly and efficiently absorbed by the lung, the skin (under conditions to prevent evaporation), and the gastrointestinal tract of humans and animals. As the duration of inhalation exposure increases, the percentage not absorbed decreases because steady-state levels are approached in the blood and tissues, and 1,1,1-TCA is metabolized at a low rate. Animal studies have demonstrated that, once absorbed, 1,1,1-TCA is distributed by the blood to tissues and organs throughout the body, including developing fetuses, with preferential distribution to fatty tissues.

The volatility of 1,1,1-TCA makes acute inhalation the most likely lethal exposure scenario in humans. The acute lethal air concentration for humans is unknown; however, simulations of several lethal exposures suggest that it may be as low as 6,000 ppm. The results of animal studies indicate that the lethal exposure concentration decreases substantially with increasing exposure duration. Human deaths after inhalation exposure have been attributed to respiratory failure secondary to central nervous system depression and cardiac arrhythmias. Very little is known about lethality due to ingestion of 1,1,1-TCA. Accidental ingestion of 600 mg/kg was not fatal. Human deaths involving dermal exposure have not been reported and it is extremely unlikely due to the high volatility of 1,1,1-TCA.

1,1,1-TCA may be a hepatotoxicant in humans, although the evidence is not conclusive. Mild hepatic changes have been found by liver biopsy in exposed individuals, and at autopsy in individuals who died after acute inhalation exposure to high concentrations of 1,1,1-TCA. Animal studies indicate that exposure to relatively high 1,1,1-TCA concentrations in air ( $\geq 1,000$  ppm) or high oral doses ( $\geq 1,334$  ppm) are required to produce liver injury.

Neurological effects are the preeminent signs of acute inhalation exposure to 1,1,1-TCA in humans. The severity of effects in humans during acute inhalation exposure increases as the exposure duration and level are increased. Impaired performance of psychophysiological function tests has been observed in individuals exposed to low concentrations ( $\geq 175$  ppm). Dizziness, lightheadedness, and loss of coordination are caused by exposure to moderate concentrations ( $> 500$  ppm). General anesthesia occurs at high levels ( $\geq 10,000$  ppm). There are no reports of irreversible neurological impairment in humans.

Evidence for or against an association between exposure to 1,1,1-TCA and cancer in humans has not been established. Among animals, no effects were found in well-designed inhalation study at exposure levels  $\leq 1,500$  ppm.

USEPA has not classified 1,1,1-TCA as a human carcinogen (Group D) due to inadequate or no evidence of carcinogenicity in animal studies. To date, USEPA has not established any toxicity values for 1,1,1-TCA. Available information on 1,1,1-TCA is currently being reviewed.

